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# Is House Dust the Missing Exposure Pathway for PBDEs? An Analysis of the Urban Fate and Human Exposure to PBDEs

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Polybrominated diphenyl ether (PBDE) body burdens in North America are 20 times that of Europeans and some "high accumulation" individuals have burdens up to 1−2 orders of magnitude higher than median values, the reasons for which are not known. We estimated emissions and fate of ΣPBDEs (minus BDE-209) in a 470 km$^2$ area of Toronto, Canada, using the Multi-media Urban Model (MUM-Fate). Using a combination of measured and modeled concentrations for indoor and outdoor air, soil, and dust plus measured concentrations in food, we estimated exposure to ΣPBDEs via soil, dust, and dietary ingestion and indoor and outdoor inhalation pathways. Fate calculations indicate that 57−85% of PBDE emissions to the outdoor environment originate from within Toronto and that the dominant removal process is advection by air to downwind locations. Inadvertent ingestion of house dust is the largest contributor to exposure of toddlers through to adults and is thus the main exposure pathway for all life stages other than the infant, including the nursing mother, who transfers PBDEs to her infant via human milk. The next major exposure pathway is dietary ingestion of animal and dairy products. Infant consumption of human milk is the largest contributor to lifetime exposure. Inadvertent ingestion of dust is the main exposure pathway for a scenario of occupational exposure in a computer recycling facility and a fish eater. Ingestion of dust can lead to almost 100-fold higher exposure than "average" for a toddler with a high dust intake rate living in a home in which PBDE concentrations are elevated.

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## Introduction

Polybrominated diphenyl ether (PBDE) concentrations in humans and the environment in North America have doubled approximately every 4−6 years or less (*1*−*3*) due to their pervasive use in North America as flame retardants. Hites (*1*) has calculated that the PBDE body burden of the typical North American is 20 times that of a typical European. Some individuals have been found to have PBDE levels in blood far above the elevated North American average, up to 1−2 orders of magnitude higher than median values (e.g., refs *1*, *4*, and *5*), for which dust is suspected to be the main contributor (*6*, *7*). Exposure assessments have shown that human milk is a major source of PBDEs to infants (*6*), but this begs the question of the source of PBDEs to nursing mothers. Typically, exposure assessments of adults have focused on the dietary exposure pathway (*8*−*10*). Few studies have examined exposure to PBDEs via inhalation (*11*) or other oral pathways such as soil and dust or dermal routes (e.g., refs *1*, *12*, and *13*). These studies fail to account for elevated exposures and body burdens as well as all possible exposure pathways (*14*).

Any strategies to limit exposure to PBDEs require an understanding of their sources and fate in the environment. The most likely urban sources for emissions of the three commercial mixtures of PBDEs (i.e., penta-BDE, octa-BDE, and deca-BDE) are from releases during their use in manufacturing commercial products (e.g., acrylonitrile−butadiene−styrene and polystyrene plastics, polyurethane foams) and releases from commercial products such as furniture, electronic equipment (e.g., computers, televisions), and small motor appliances (e.g., hair-dryers) during their use and during subsequent disposal (*15*). For releases from in-use products, PBDEs are emitted indoors and then move outdoors (*16*, *17*) where they are subject to environmental fate processes. The fate processes include regional and long-range transport (*18*, *19*) that, due to their persistence, result in PBDEs being transferred to ecosystems and agricultural food systems. This process of release and environmental transport is presumably analogous to historical releases of polychlorinated biphenyls (PCBs), which although they were never manufactured in Canada, were released in part during their use indoors in, for example, window caulking, light ballasts, and carbonless copy paper (e.g., refs *20* and *21*). However, as Betts (*14*) has pointed out, the current situation of the release of PBDEs from ubiquitous sources in homes and workplaces distinguishes PBDEs from "legacy" POPs such as PCBs and polychlorinated dibenzodioxins and furans (PCDD/F) for which past releases have resulted in their current worldwide distribution.

Both regulatory agencies and private industry have moved toward limiting the manufacturing and use of PBDEs. Canada has proposed the "virtual elimination" of tetra-, penta-, and hexa-BDE congeners and implementation of controls for hepta- through deca-BDE congeners (*22*). It is likely that consumer products will continue to be a source of PBDEs to the environment, particularly the indoor environment in the near future. Since Canadians spend most of their time indoors (*23*, *24*), indoor exposure pathways are thought to contribute a significant amount of PBDEs to the North American daily intake (*1*, *7*).

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**TABLE 1. Physical−Chemical Properties Used in the MUM-Fate Model**

| | media | | | | | |
|---|---|---|---|---|---|---|
| | air (upper) | air (lower) | soil | water | veg'n | film |
| surface area ($m^2$)[a] | $4.69 \times 10^8$ | $4.69 \times 10^8$ | $4.69 \times 10^7$ | $1.17 \times 10^8$ | $5.63 \times 10^7$ | $6.90 \times 10^8$ |
| depth (m)[b] | 450 | 50 | 0.02 | 10 | 0.002 | $10^{-7}$ |
| volume ($m^3$)[b] | $2.11 \times 10^{11}$ | $2.35 \times 10^{10}$ | $9.39 \times 10^5$ | $1.17 \times 10^9$ | $1.13 \times 10^4$ | 69.0 |
| $v_A$[b] | ~1 | ~1 | 0.2 | | 0.8 | |
| $v_W$[b] | | | 0.3 | ~1 | 0.15 | |
| $v_{Part}$[b] | $6.7 \times 10^{-11}$ | $6.7 \times 10^{-11}$ | - | $3 \times 10^{-5}$ | - | 0.88 |
| $v_{Organic\ carbon}$[b] | | | 0.06 | | 0.05 | 0.12 |
| advective flow ($m^3$/h)[c] | $2.9 \times 10^{11}$ | $1.6 \times 10^{10}$ | | $3 \times 10^8$ | | |
| advective residence time (h)[c] | 0.7 | 1.4 | | 4.7 | | |
| reaction half lives (h)[d] | | | | | | |
| BDE-17 | 180 | 180 | 1500 | 1500 | 130 | 100 |
| BDE-28 | 180 | 180 | 1500 | 1500 | 130 | 100 |
| BDE-47 | 260 | 260 | 10000 | 10000 | 180 | 130 |
| BDE-66 | 250 | 250 | 10000 | 10000 | 180 | 130 |
| BDE-77 | 250 | 250 | 10000 | 10000 | 180 | 130 |
| BDE-85 | 470 | 470 | 30000 | 30000 | 330 | 240 |
| BDE-99 | 470 | 470 | 30000 | 30000 | 330 | 240 |
| BDE-100 | 360 | 360 | 30000 | 30000 | 250 | 190 |
| BDE-126 | 370 | 370 | 30000 | 30000 | 260 | 190 |
| BDE-153 | 1110 | 1110 | 87600 | 87600 | 780 | 580 |
| BDE-154 | 690 | 690 | 87600 | 87600 | 490 | 360 |
| BDE-183 | 850 | 850 | 87600 | 87600 | 600 | 450 |

[a] Ref *29*. [b] Ref *28*. [c] Ref *33*. [d] Estimated from AOPWIN and BIOWIN, software supported by U.S. EPA and converted to first-order degradation rates following approach in refs *28* and *36*.

Measured body burdens are typically higher than expected from dietary intake estimates (e.g., ref *25*), leading researchers (*26*) to search for (i) missing exposure pathways of significance; (ii) reasons why exposures in some individuals, who are referred to as "super-accumulators", are much higher than mean or median exposures; or (iii) variable PBDE pharmacokinetic mechanisms possibly caused by genetic polymorphisms. Our work addresses the "missing exposure pathway" question by considering two exposure pathways that have not been well-characterized, namely, indoor inhalation and ingestion of household dust. Our work also explores additional dietary exposure pathways related to fruit, vegetable, and grain products that have not been considered in PBDE exposure assessments. We address variability among individuals by quantifying exposures from several scenarios that may be applicable to particular sub-populations.

This paper (i) estimates the emissions and fate of ΣPBDEs (minus BDE-209) in an urban area; (ii) quantifies the exposure to ΣPBDEs of average urban Canadians; (iii) investigates possible exposure pathways and scenarios that could account for high levels of PBDEs in some individuals; and (iv) addresses the issue of how nursing mothers could be acquiring the PBDEs that they subsequently pass on to their infants via human milk. Our aim is to contribute knowledge that may assist in developing strategies to reduce human exposures to PBDEs.

## Materials and Methods

**Chemical Fate.** MUM-Fate, a steady-state (level III), non-equilibrium, fugacity-based multimedia chemical fate model (*27*, *28*) was used to estimate emissions and media concentrations in an urban environment. MUM-Fate, which considers six compartments (i.e., air, soil, vegetation, surface film, water, and sediment), was parameterized for a 470 $km^2$ area centered on Toronto, Canada. Surface area coverage was estimated through analysis of land-use maps (*29*). Approximately 1.3 million people live in this area, which has 60% impervious surface coverage with an Impervious Surface Index of 2.3 (*28*) and 10% coverage by vegetation with a Leaf Area Index of 1.6 (*30*). The water compartment is primarily nearshore Lake Ontario that covers approximately 30% of the modeled domain. The previous version of MUM-Fate (*27*, *28*) treated air as completely mixed from ground surface to boundary layer height and likely underestimated the chemical mass emitted near urban surfaces that interacted with these surfaces. The version presented here includes lower (0−50 m) and upper (50−500 m) air compartments and differentiates the fate of chemical loadings through emissions within the modeled domain (e.g., from indoor sources or industrial uses) from those advected from upwind sources. The 50 m lower mixing area was based on the depth of the mechanically well-mixed layer for compact residential, industrial, and <10-story commercial land-use categories (*31*) with an upper−lower air box exchange rate of 0.5 m/s, which is in the range of vertical wind speeds reported in urban canyons (*32*), with an average horizontal wind speed of approximately 4 m/s (*33*). Table 1 summarizes compartment dimensions and other model input data. The reader is referred to ref *28* for a complete explanation and a listing of all parameter values used.

The model considered a summer scenario at 25 °C using an average rain rate (*33*). The model was run for each of 10 PBDE congeners, and the results are summed to yield ΣPBDE. BDE-209 was not included for consistency with the exposure assessment. Although BDE-209 is explicitly excluded from the estimates, some BDE-209 is implicitly included through debromination to lower brominated congeners (*34*). The connection between BDE-209 and lower brominated congeners merits further attention as the transformation pathways are clarified, since ~45% of the deca formulation is consumed in North America, which comprises ~75% of our total use (*35*).

Physical−chemical data for the PBDE congeners (Table 1) were the internally consistent parameters reported by Wania and Dugani (*19*). Qualitative environmental degradation half-lives in all media were obtained by the U.S. EPA's AOPWIN and BIOWIN programs; they were converted to quantitative degradation rates as described by Diamond et al. (*28*) and Gouin and Harner (*36*). This approach does not explicitly account for the formation of lower brominated congeners due to debromination of higher brominated congeners (*34*). Similarly, this approach provides degradation

**TABLE 2. Comparison of Measured and Modeled Concentrations Obtained from MUM-Fate**

| | selected North American measured (urban, suburban, and rural) | | | modeled (urban) |
|---|---|---|---|---|
| medium | location; year | total PBDEs | ref | total PBDEs |
| air | Ontario; 2000 | 10−1300 pg/m$^3$ (gas & particle) | *36* | 45−109 pg/m$^3$ (gas & particle) |
| | Ontario; 2000 | 3.4−46 pg/m$^3$ (gas) | *37* | |
| indoor dust | United States | <DL−35.7 $\mu$g/g | *43* | 1.6−105 $\mu$g/g |
| | Washington, DC | 0.780−30.1 $\mu$g/g | *7* | |
| soil | United States; 2000 | <0.1−76 $\mu$g/kg dw | *66* | 2.0−4.8 $\mu$g/kg |
| water | Lake Ontario; 1999 | 6 pg/L | *67* | 0.9−2 pg/L[a] |
| surface films | Toronto; 2002 | 0.5−28.1 ng/m$^2$ | *16* | 7.2−17 ng/m$^2$ |

**TABLE 3. Summary of Concentrations, Sources, and Type of Data Used for the Exposure Assessment of All Life Stages and Scenarios[a]**

| | | exposure scenario | | | |
|---|---|---|---|---|---|
| media | exposure pathway | average urban Canadian | occupational | fish eater | elevated indoor source(s) |
| air (pg/m$^3$) | indoor home | 4.45 × 10$^2$ (*41*); mean | 4.45 × 10$^2$ (*41*); mean | 4.45 × 10$^2$ (*41*); mean | 7.29 × 10$^3$ (*41*); max |
| | indoor at work | na[b] | 1.64 × 10$^3$ estimated from (*16*); single value | na[b] | na[b] |
| | outdoor | 8.18 × 10$^1$ (*37*); mean | 8.18 × 10$^1$ (*37*); mean | 8.18 × 10$^1$ (*37*); mean | 8.18 × 10$^1$ (*37*); mean |
| soil ($\mu$g/kg dry wt) | outdoor | 3.09 × 10$^0$ MUM-Fate; mean | 3.09 × 10$^0$ MUM-Fate; mean | 3.09 × 10$^0$ MUM-Fate; mean | 3.09 × 10$^0$ MUM-Fate; mean |
| dust ($\mu$g/g) | indoor | 5.06 × 10$^0$ estimated from ref *41*, mean | 5.06 × 10$^0$ estimated from ref *41*, mean | 5.06 × 10$^0$ estimated from ref *41*, mean | 1.05 × 10$^2$ estimated from ref *41*, max |
| | work | na[b] | 3.02 × 10$^1$ estimated from ref *16*; single value | na[b] | na[b] |
| food (ng/kg fresh wt) | dairy, meat and eggs | 101 (*44, 45*); mean | 101 (*44, 45*); mean | 101 (*44, 45*); mean | 101 (*44, 45*); mean |
| | fish and shellfish | 417 (*44, 45*); mean | 417 (*44, 45*); mean | 417 (*44, 45*); mean | 417 (*44, 45*); mean |
| | plant products | 23 (*8, 46*); mean | 23 (*8, 46*); mean | 23 (*8, 46*); mean | 23 (*8, 46*); mean |
| | fats, nuts and oils | 81 (*44, 45*) mean | 81 (*44, 45*) mean | 81 (*44, 45*) mean | 81 (*44, 45*) mean |
| | sugars and sweets | 190 (*44, 45*) mean | 190 (*44, 45*) mean | 190 (*44, 45*) mean | 190 (*44, 45*) mean |
| | human milk (whole milk, 4% lipid) | 2400 (*47*); mean 32−38240 (*47*); min−max | | 2400 (*47*); mean | 2400 (*47*); mean |

[a] Italized numbers refer to references. [b] Exposure pathway not applicable

rates that decrease with increasing bromination, which is the converse of the observations made regarding photolytic degradation pathways for PBDEs.

Concentrations of each PBDE [includes BDE-17, -28, -47, -66, -77, -85, -99, -100, -126, -153, -154, and -183] congener advected into the modeled domain were derived from results for passive air samplers deployed in three suburban and rural sites north of Toronto by Harner et al. (*37*). Passive air sampler data were assumed to represent gas-phase concentrations only; therefore, particulate-phase PBDE were estimated based on the $K_p$−$K_{OA}$ relationship of Finizio et al. (*38*). Using a rural total suspended particulate concentration of 15 $\mu$g/m$^3$, gas- and particulate-phase concentrations were summed for the estimate of total input air concentrations. Total urban air concentrations were calculated in the same manner from five Toronto urban sites, also from Harner et al. (*37*), using an urban total suspended particulate concentration of 80 $\mu$g/m$^3$. A range of likely emissions within the 470 km$^2$ area was then calculated as the input required, in addition to the rural/suburban advection, for MUM-Fate to achieve an advected air output that equaled the Toronto urban air concentrations. In all, six emission scenarios for ΣPBDE were derived from three advection scenarios and two urban air output scenarios. The ranges of modeled concentrations in soil and film were compared with literature values to evaluate model efficacy (Table 2).

Exposure to ΣPBDEs was estimated using the exposure module of a generic multimedia human health risk assessment model, MUM-FAMrisk (*39*). Exposure was calculated for an "average urban Canadian" adult (20+ years) and for adults in three additional scenarios intended to explore potentially elevated exposures: (a) elevated indoor source(s); (b) fish eater; and (c) occupational exposure, specifically from an electronics recycling facility located in the Greater Toronto Area (*16*). We also estimated the "average" exposure of four younger age classes including infant (0−6 months), toddler (6 months−4 years), child (5−11 years), and teen (12−19 years), as defined by Health Canada (*40*) and explored the range of exposures obtained from ingesting low and high concentrations of human milk (infant) and dust (toddler and adult). We used a combination of measured and modeled concentrations for environmental media and only measured concentrations for food (Table 3). We used ΣPBDE rather than specific congeners because many of the data were expressed as ΣPBDE. All ΣPBDE from various sources contained BDE-47, -99, -100, -153, and -154. As mentioned above, BDE-209 was excluded from the analysis due to a lack of data in most media. As such, our fate and exposure calculations underestimate total values.

Modeled data used in the exposure assessment were ΣPBDE in outdoor soil, outdoor air (gas- and particle-phase), indoor air (gas- and particle-phase), and dust in the residential [includes BDE-17, -28, -47, -66, -71, -85, -99, -100, -153, and -154] and occupational scenarios [includes BDE-17, -28, -47, -66, -77, -85, -99, -100, -126, -153, -154, and -183]. Mean and maximum measured concentrations of gas-phase ∑PBDEs in residential indoor air were taken from Wilford et al. (*41*), who deployed passive samplers in 94 homes in Ottawa, Canada, a city of similar socio-economic status to Toronto but with a lower population. ∑PBDEs in the occupational setting's indoor air were taken from Butt et al. (*16*), who estimated gas-phase air concentrations from films collected on impervious surfaces (i.e., windows) at an electronics recycling facility where computers are dismantled. Particle-

phase concentrations in air were calculated from gas-phase concentrations using the equilibrium $K_p-K_{OA}$ relationship of Finizio et al. (*38*), assuming a room temperature of 21 °C. ∑PBDE minimum, mean, and maximum concentrations in residential house dust were similarly calculated from measured gas-phase indoor air concentrations (*41*) using the same equilibrium $K_p-K_{OA}$ relationship and using physical properties for dust reported by Bennett and Furtaw (*42*). The modeled concentrations reported in Table 2 fall within the range of reported values (e.g., refs *7* and *43*).

Arithmetic mean concentrations of ∑PBDEs in fatty foods [included BDE-15, -17, -28, -47, -66, -71, -75, -77, -85, -99, -100, -119, -126, -138, -153, -154, -183, and -190] were taken from market basket surveys conducted in two Canadian cities, Vancouver (*44*) and Whitehorse (*45*). ∑PBDE concentrations in non-fatty foods (fruit, vegetables, and grain) were arithmetic mean concentrations from recent studies from Finland [included BDE-47, -99, -100, -153, and -154] (*8*) and Ottawa, Canada [included BDE-15, -17, -28, -47, -66, -71, -75, -77, -85, -99, -100, -119, -126, -138, -153, -154, -183, and -190] (*46*). The mean, maximum, and minimum concentrations of ∑PBDE in Canadian human whole milk [included BDE-28, -47, -99, -100, -153, -154, and -183] were calculated as the sum of congener-specific measurements from 98 whole milk samples collected across Canada in 2002 (*47*).

Canadian food and media intake rates for all life stages were taken from Nutrition Canada Survey data of Health Canada (*40*). Use of dietary survey information based solely on data generated for consumers only (zeros out) would tend to overestimate intake rates for nonconsumers, and for this reason primary data used to estimate mean Canadian exposures included both consumers and nonconsumers (zeros in).

Exposure was calculated for each media and food pathway as:

$$\text{Media}_{exp} = \frac{\text{IR}_{media} \times \text{BIO} \times \text{Media}_{concn} \times \text{Fr}_{day}}{\text{BW}} \quad (1)$$

where $\text{Media}_{exp}$ is the contaminant exposure estimate through one medium ($ng/kg_{body\ wt}\cdot day$), $\text{IR}_{media}$ is the media intake rate (kg/day), BIO is the bioavailability of the contaminant in that medium (dimensionless; assumed alternately as 100% and 50%; see text below for explanation), $\text{Media}_{concn}$ is ∑PBDE concentration of the medium (ng/kg), $\text{Fr}_{day}$ is the fraction of time spent exposed to the medium during the day (dimensionless), and BW is body weight (kg). $\text{Media}_{exp}$ were summed to yield an estimated daily intake (EDI) (ng/day). Congener values below the detection limit were assumed to equal zero.

Bioavailability (absorption) factors of 100% and 50% from the GI tract were assumed for purposes of our calculations. A value of 100% is consistent with the implicit assumption employed by Stapleton et al. (*7*) in their calculation of potential doses to children as a result of ingesting PBDE-contaminated house dust in homes in Washington, DC. The systemic absorption of 2,2′,4,4′-tetrabromodiphenyl ether delivered in corn oil exceeded 80% in mice (*48*). However, there are no specific data or information currently published on the in vivo mammalian bioavailability or in vitro bioaccessibility of PBDEs from soil or house dust. A common web-based risk assessment tool employed for contaminated site risk assessment—the Risk Assessment Information System (*49*)—provides recommended gastrointestinal absorption factors for soil-borne contaminants. For mono-, di-, tri-, penta-, and octa-bromodiphenyl ethers, RAIS recommends an assumption of 50% GI absorption. There is no explanation or rationale provided to support this recommended absorption factor. However, to evaluate the implications of reduced bioavailability of PBDEs on house dust, we have estimated intakes from ingestion of dust assuming scenarios of both 100% and 50% GI absorption.

Canadian time-activity data from Richardson (*23*) were averaged over 12 months for Canadians over the age of 11 years, while those data for children under age 11 (infants, toddlers, children), were derived from yearly average time–activity data based on the Canadian Human Activity Pattern Survey (CHAPS) (*24*). Soil/dust ingestion exposures were calculated on a daily basis, assuming that a receptor spends 92−>94% of time indoors where they are assumed to be exposed only to dust, and the remaining time is spent outdoors where they are exposed only to soil. The average soil/dust ingestion rates vary from 0.05 to 0.02 g/day for toddlers and teen/adults, respectively (*40*). Inhaled dust and resuspended dust are accounted for through the inhalation pathway, which includes gas and particle phases.

As mentioned above, we developed several likely exposure scenarios based on available data, the "elevated indoor source(s)" (all age classes) (scenario a) were assumed to live in a home with high levels of PBDEs, which could be due to household items (e.g. electronic and electrical equipment, insulation, soft furnishings, curtains and bed mattresses) or poor home ventilation. Exposure for the fish eater (scenario b) was assessed using the adult fish and shellfish intake rate of Richardson (*23*). Fish and shellfish concentrations used were the arithmetic means of marine fish, freshwater fish, canned fish, and shellfish concentrations from Vancouver, 2002 (*44*), which are greater than those measured from Whitehorse (*45*). The selection of these four different sources of fish were consistent with those selected in total diet studies intended to estimate exposures for fish eaters among the general public who purchase fish from the supermarket. Since we used the mean intake rate of Richardson (*23*) that is for eaters only (rather than a maximum fish and shellfish intake rate) and the higher average for fish of the two measured diet surveys, this scenario approximates a reasonable but not extreme maximum exposure. The occupational exposure (scenario c) was assumed to occur 40 h per week throughout the year at an electronics recycling facility.

## Results and Discussion

**Chemical Fate Model Results.** MUM-Fate results during this summer scenario provide emissions of 100−422 g/day of ∑PBDEs to the 470 $km^2$ model area, compared with an average of 74−207 g/day of PBDEs from upwind sources outside the modeled domain (Figure 1). These estimated emission rates result in media concentrations that are within an order of magnitude of literature values (Table 2, Supporting Information Table 1). Modeled air concentrations were forced to equal measured air concentrations (*37*) as a result of the emission rate selected. Soil concentrations are within the measured range reported; however, these measured values were downwind of a polyurethane foam manufacturer and span a range from below a detection limit that is an order of magnitude below modeled concentrations to an order of magnitude above modeled concentrations. Water, as a mobile medium, was assumed to have an input at a concentration of zero, thus the modeled concentrations represent net increase above urban background. Although there is substantial uncertainty in estimates of total emissions, the result that 57−85% of ∑PBDE originate from within Toronto (rather than advection from regional inputs) is consistent with the congregation of likely residential and small industrial emission sources within the city (*50*). The range of emissions derived for BDE-47 and -99 for Toronto corresponds to ∼19−82 mg $capita^{-1}$ $year^{-1}$ in the modeled area, which are ∼3−12 times greater than the 7.0 mg $capita^{-1}$ $year^{-1}$ for Swedish citizens estimated by Palm (*51*) based on a substance flow analysis for Denmark. Given that North American concentrations are 20-fold higher than those of Europe and that the

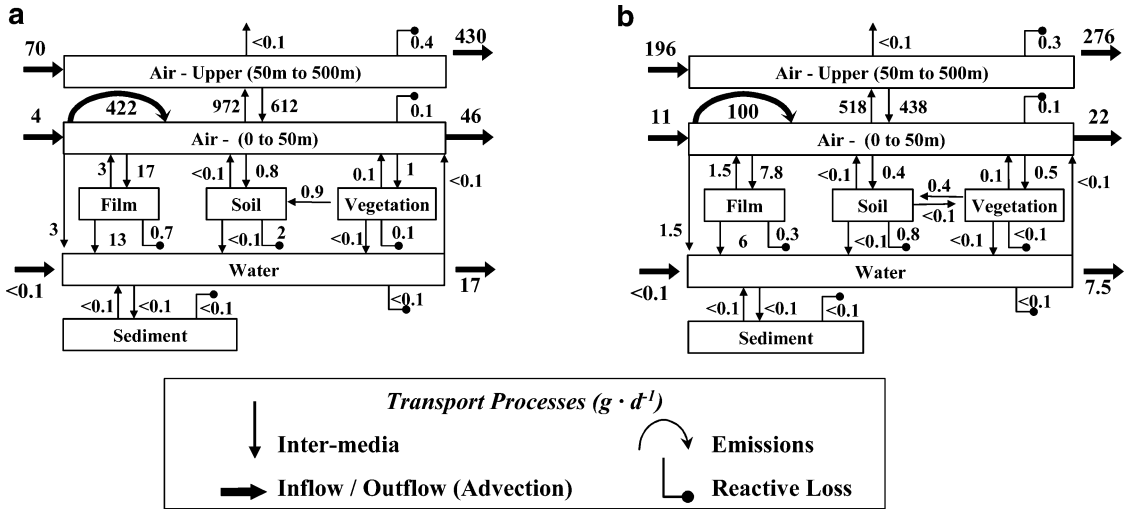


**FIGURE 1. Fate of EPBDEs estimated by MUM-Fate: (a) minimum concentration of 10 pg/m$^3$ in advective air input and the maximum advective air output concentration of 109 pg/m$^3$; (b) maximum concentration of 28 pg/m$^3$ in advective air input and the minimum advective air output concentration of 45 pg/m$^3$. Rates (expressed as g/day) were obtained by running the model for each congener and then summing the results. Model was parameterized to resemble Toronto, Canada, and range of advective air concentrations was obtained from ref *37*. Congeners included are listed in Table 1.**

estimates from Palm (*51*) and this paper were obtained using very different methods, the difference in estimates is not surprising.

Over 95% of ΣPBDE entering the model domain are lost through air advection. The remaining emissions undergo either degradation or multi-media partitioning and result in increased urban media concentrations (Figure 1, Table 2). Approximately 5% (8−17 g/day) of emissions are transferred to films on impervious surfaces that achieve the highest concentration among media of 121−292 ng/g or 7.2−17 ng/m$^2$. In turn, 1.5−3 g/day volatilizes from the films, and 6−13 g/day is transferred to surface water (near-shore Lake Ontario), resulting in concentrations of up to 2 pg/L, where it is available for uptake into the aquatic food web. Modeled concentrations of the various congeners in the upper air box (50−500 m) varied from 27 to 81% of the lower air box (0−50 m) values, which agrees reasonably with the data presented by Harner et al. (*37*) from passive samplers deployed at Toronto's CN tower where they found congener concentrations at 300 m were 14−54% of the 30 m congener concentrations.

There are numerous sources of uncertainty associated with estimates of emissions and fate. Diamond et al. (*28*) discuss uncertainties and limitations of the MUM-fate model. Specific to this application, the major uncertainties are exclusion of BDE-209 from the calculations, process rates in general, and the back-calculation of within city emissions from regional air inputs. The uncertainty in the latter is, in part, attributable to variations of 3−4 times within each of the urban and the suburban/rural samples and that while urban concentrations are on average 2−3 times higher than suburban/rural measurement from the same time frame, periodically a congener in the urban environment can be at a lower concentration than in the suburban/rural environment (*37*). Degradation rates and the potential for transformation of higher into lower brominated congeners are also major uncertainties in the model, particularly since the rates used were estimated and not measured values, with congener-specific reaction rates decreasing with increasing bromination. Due to the steady-state nature of the model, we have ignored seasonal and diurnal variations in degradation and transport rates (e.g., temperature, wind speed and direction, precipitation, atmospheric mixing height, etc.). As illustration of this effect, considering a temperature of 0 rather than 25 °C increases partitioning to solid phases, such as soils and surface waters (e.g., increasing BDE-99 concentrations 2.5 times in water and 24 times in soil).

**Exposure for Different Life Stages.** Estimated daily intakes of ΣPBDE for the average urban Canadian (all age classes) ranged from 155 ng/day for the adult to 1965 ng/day for the breast-fed infant (Figure 2a). This corresponds to 2 and 280 ng/kg$_{body\ wt}$·day for the adult and infant, respectively. Human milk contributed most to the infant's exposure. The importance of human milk to lifetime exposure is not surprising, given the relatively high PBDE concentrations reported in North American human milk as compared to other foods. For example, Ryan (*47*) reported Canadian human milk ∑PBDE concentrations on a whole milk basis assuming 4% milk fat (ranging from 32 to 38 240 ng/kg; arithmetic mean and median are 2400 and 880 ng/kg whole milk; $n$ = 98). These concentrations are up to 10-fold higher than those reported in other foods (ranging from "no detects" to 1190 ng/kg wet weight) (*52*). Schecter et al. (*53*) also reported high ∑PBDE concentrations in human milk from American women (ranging from 248 to 16 800 ng/kg whole milk; $n$ = 47). Using the range in ΣPBDE concentrations for Canadian women's milk (*47*), the intake rate via human milk for Canadian infants could vary from 24 to 28 680 ng/day or 3.4−4100 ng/kg$_{body\ wt}$· day, for the low and high concentrations, respectively (Figure 2b).

The high concentrations in human milk beg the question of the source(s) of PBDEs to the nursing mother and other life stages. We found that house dust contributed most to exposures for all the other life stages, where the adult life stage includes the nursing mother (Figure 2a). Our results are substantiated by several studies (*6*, *7*, *54*). For all life stages, high exposures are a product of both high concentrations in this medium and spending 22 out of 24 h per day indoors (*23*, *24*). High ΣPBDE concentrations in house dust are attributable to the numerous emission sources within the indoor environment as well as the high organic carbon content of approximately 19% expected for house dust (*42*). Buildings with poor ventilation can also achieve high concentrations as degassed PBDEs accumulate in the indoor environment. Humans are exposed to house dust through several exposure pathways, namely, direct inhalation of resuspended dust; ingestion of dust through direct and indirect pathways; and, dermal exposure on the hands and body (although we neglect the latter due to high uncertainties and presumed low rates of dermal transfer). Our exposure

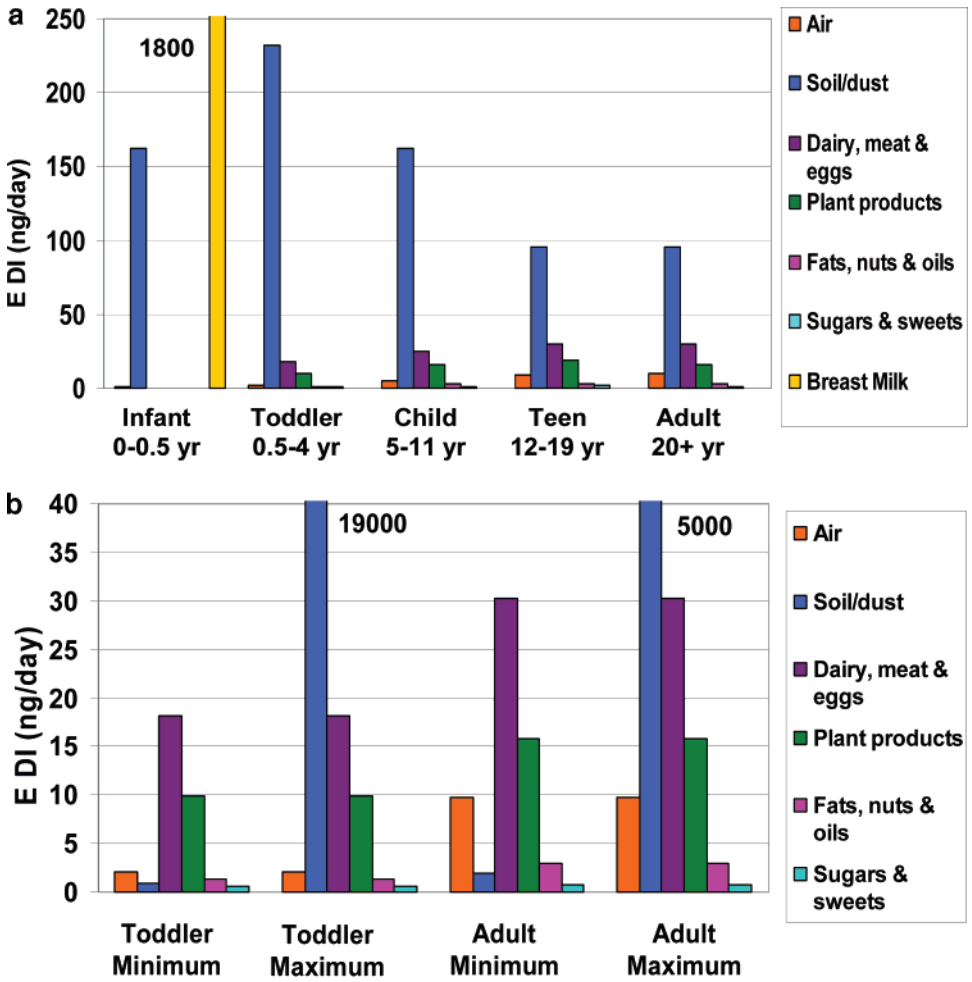


**FIGURE 2. Exposure pathway contributions to estimated daily intake (EDI, ng/day) of ΣPBDE for (a) different life stages of an average urban Canadian and (b) minimum and maximum available soil/dust intake rates for toddlers and adults and minimum and maximum estimated concentrations. Main congeners considered were BDE-47, -99, -100, -153, and -154; however, particular exposure pathways may have included additional congeners (see text).**

estimates for dust indicate that ingestion is the critical exposure pathway, with dust inhalation (via particle phase air concentrations) contributing minimally. However, to clarify this point, ingestion includes the fraction of dust that is inhaled but removed from the nasal passages where upon it enters the alimentary track via mucus ingestion (a process called mucociliary clearance).

Using this "average" scenario, toddlers have particularly high exposure to house dust: dust accounts for 90% of their EDI of 264 ng/day or 20 ng/$kg_{body\ wt}$·day (Supporting Information Table 2). This result is not only due to the high PBDE concentration in dust and time spent indoors, but also the relatively high soil/dust ingestion rate of 0.05 g/day (*40*). ΣPBDE intake via dust could, however, range from 0.46 to 19,270 ng/day and 0.035−1482 ng/$kg_{body\ wt}$·day if using the low and high soil/dust intakes of 0.01−0.2 g/day from the U.S. EPA (*55*), in combination with the 5th percentile and maximum estimated dust concentrations of 0.1−105 $\mu$g/g (Figure 2b) and estimates of bioavailability of 50 and 100%. The range of dust intakes accounts for 1.4−99.8% of the toddler's EDI. In comparison, the adult's range of dust intakes is 0.8−4970 ng/day or 0.02−71 ng/$kg_{body\ wt}$·day using Health Canada's soil/dust intake rate of 0.02 g/day (*40*) and the U.S. EPA value of 0.05 g/day (*56*), minimum and maximum dust concentrations (Figure 2b), and bioavailabilities of 50 and 100%.

The high exposure of toddlers and even infants via dust is consistent with assessments of lead exposure where children's blood lead levels have been positively correlated with lead concentrations in house dust (e.g., ref *57*). Toddlers are apt to ingest dust due to their frequent hand-mouth contact and "mouthing" toys and other objects in contact with floors. However, our estimated dust exposure rates contain considerable uncertainty because, although the dust intake rates used here have been well vetted, the rates are based on a limited number studies, most of which dealt with the intake of soil and not dust (*55*).

Another source of uncertainty is our assumption of gastrointestinal bioavailability, estimates of which are not available for humans in any medium. Indoor dust has a significantly greater organic carbon content and smaller particle size relative to outdoor soils (percent organic carbon of 20−40% in dust versus 2−5% common for surface soils) (*58*). Hence, dust bioavailability may be reduced compared to estimates for soils, and is an important consideration in exposure modeling. Data on ingestion bioavailability for mammals is extremely limited and nonexistent for absorption from ingested soil or dust. Some 80% of PBDE in oil may be absorbed from the mammalian GIT (*48*). GIT absorption in fish ranges between 40% and 95% (decreasing with increasing bromination; reviewed by Sjodin et al. (*59*)), but the relevance of these data to humans or other mammals is unknown at the present time. As a result, we selected bioavailabilities representing (a) the maximum absorption scenario and (b) a lower estimate to investigate the effects of reduced bioavailability.

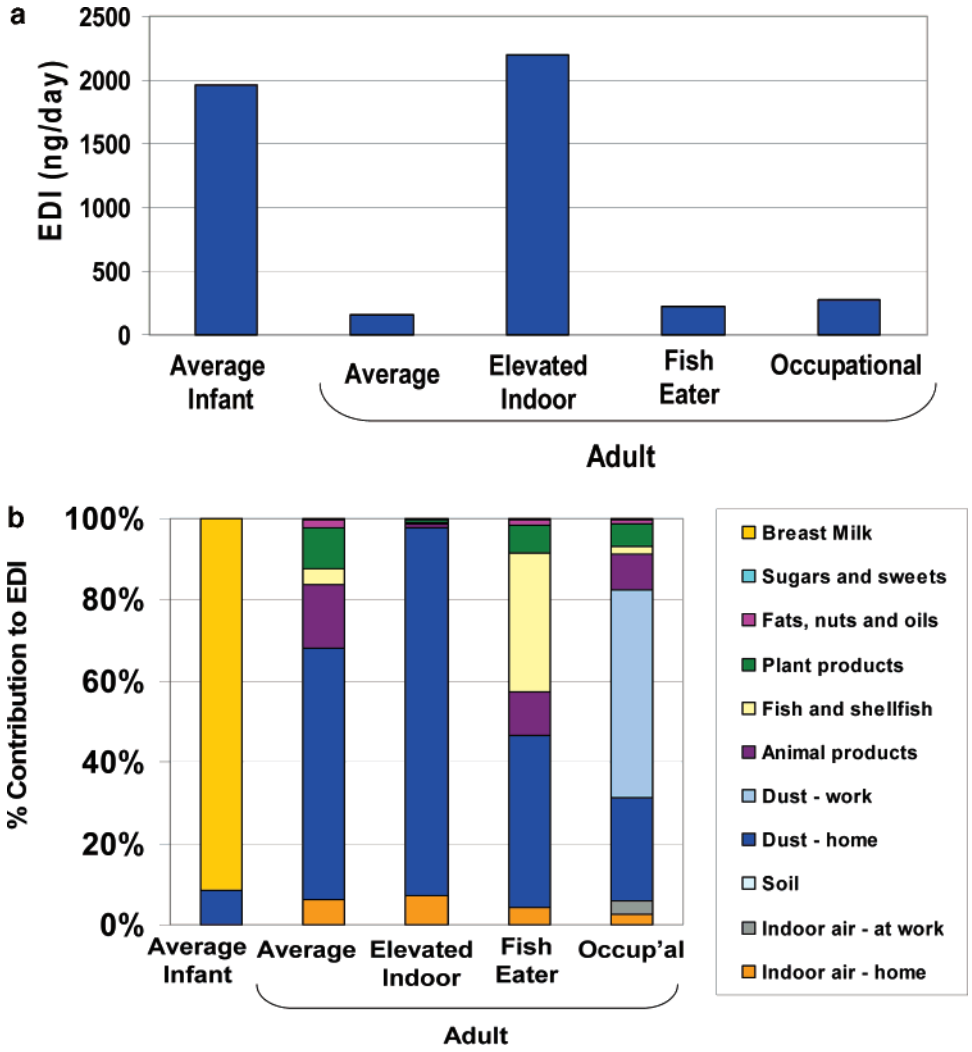


**FIGURE 3. Results of exposure scenarios for ΣPBDE for (a) estimated daily intakes (EDI, ng/day) for each scenario and (b) percentage contributions of exposure pathway to EDIs. Main congeners considered were BDE-47, -99, -100, -153, and -154; however, particular exposure pathways may have included additional congeners (see text).**

In terms of the accuracy of our estimated ΣPBDE dust concentrations, the median concentration of 1.6 $\mu$g/g and the 95th percentile concentration of 25 $\mu$g/g bound the range of observed values (Table 2). The highest value of 105 $\mu$g/g exceeds reported values by a factor of 3 (*7*, *43*). The accuracy of the $K_P$:$K_{OA}$ relationship for estimating household dust concentrations from indoor air concentrations has recently been confirmed by Shoeib et al. (*60*) for suspended household dust and suggests near equilibrium conditions between these two media. Surface dust may achieve even higher concentrations relative to indoor air due to its proximity to near-surface sources. Characterization of the sources and rates of PBDE emissions, in particular from household items such as carpet underpadding, furniture, and electronics equipment, will help explain the ranges of PBDE concentrations in dust.

Following household dust, "dairy, meat, and eggs" were the next most important exposure media for the average urban Canadian (16% contribution), the elevated indoor source(s) and the occupational adult scenarios (Figures 2b and 3b). For the average urban Canadian adult scenario, beef, pork, eggs, milk and dairy products, and poultry contributed 5, 4.5, 4.3, 1.3, and 0.8% of the total daily intake of ΣPBDEs, respectively (Supporting Information Table 2). Inhalation of indoor air contributed 6% for the average urban Canadian adult. Contributions from fish were only 3.6% of total exposure, noting that this intake rate (as well as all others for food) dates back to the late 1970s when Canadian's consumed less fish that they do today. The difference in contributions from fish and other dietary sources relative to that of Kiviranta et al. (*8*) for Finland is most likely due to a combination of lower average Canadian fish intake rates and lower average fish concentrations from Canadian supermarkets than those in Finland. The risk assessment performed by VCCEP (*13*) assumed higher fish and lower dust concentrations than this study. This suggests that the relative importance of PBDE exposure pathways for the average consumer could differ geographically at a broad scale.

The importance of foods from plants is often overlooked (e.g., ref *25*), as researchers assume that, as for other persistent and bioaccumulative organics such as PCBs and PCDD/Fs, dietary contributions from animal products dominate exposures. We found that plant products contributed 10% of total dietary exposure or about 64% that of dairy, meat and eggs.

**Exposure Scenarios.** Several exposure scenarios were constructed to explore likely ranges in estimated daily intake values and to further examine the dominant exposure pathways (Figure 3a,b). Relative to the "base case" exposure of 155 ng/day of the average urban Canadian adult, exposures ranged from 227 for the fish eater scenario to 2190 ng/day for the elevated indoor source(s) scenario. Occupational (282 ng/day) was comparable to the fish eater. Ingestion of dust contributed most to exposure in the elevated indoor source(s), fish eater, and occupational scenarios.

Exposure to indoor dust in the elevated indoor source(s) and occupational exposure scenarios clearly illustrates the importance of this medium to total exposure and provides an explanation for the "missing" exposure pathway leading some individuals to be "super-accumulators". In the case of occupational exposure, the emission source is assumed to be the constant stream of electronics dismantled at the recycling facility. These results are supported by those obtained by Sjodin et al. (*54*) and Jakobsson et al. (*61*), who measured PBDEs in the blood plasma of Swedish workers exposed to computers and at an electronics recycling facility, respectively. The elevated indoor source(s) scenario differs from the occupational exposure since the release of PBDEs causing the elevated dust concentration could be due to an episodic or intermittent activity (e.g., use of a heat gun) or may decrease with time as the source item (e.g. carpet, furniture) ages (*62*). In these cases, the scenario could represent a short-term exposure. Alternatively, releases could continue as PBDE-impregnated products are continually replaced in the home or products such as foams crumble with age and possibly increase their release rate. The time-course over which the concentrations vary also depends on the air exchange rate and cleaning practices that remove dust. Since PBDEs depurate relatively rapidly following a reduction in exposure (*54*), super-accumulators might be more accurately termed "super-exposures" since the duration of elevated body burdens may be short term. This relatively rapid clearance rate and the potential to modify one's environment to reduce exposure to PBDEs contrasts with legacy POPs for which exposures are more difficult to manage because of the latter's pervasive distribution in the environment and food supply.

Fish and shellfish were the second most important exposure medium for the fish eater, which reflects our choice of the highest average fish and shellfish concentration from the available Canadian total diet survey results. This scenario approximates an upper estimate of exposure for the average Canadian fish eater. The PBDE fish concentration is derived from a market basket survey that includes both marine and freshwater fish (*44*, *45*). This exposure could be greater if fish from more highly contaminated areas were preferentially consumed (e.g., ref *63*).

Due to a lack of data, we have omitted air and dust exposure in automobiles as another potential route of exposure (*64*). It is possible that PBDEs achieve elevated concentrations inside automobiles given their abundance in the polyurethane foam seats and potentially high temperatures that promote degassing.

**Implications.** The risks posed by these estimated exposures are difficult to characterize. To our knowledge, only the U.S. EPA has attempted to establish reference exposure levels for PBDEs, having derived chronic oral reference doses (RfD) for three commercial mixtures: penta (RfD = 0.002 mg/kg$_{body\ wt}$·day); octa (RfD = 0.003 mg/kg$_{body\ wt}$·day); and deca (RfD = 0.01 mg/kg$_{body\ wt}$·day) (*65*). Toxicological endpoints considered include effects related to the liver and exclude the potentially more sensitive health effects in humans related to embryonic, fetal, infant and child development, and reproduction. Furthermore, the current toxicological literature on PBDEs is lacking in human health studies, and information must be gathered regarding the timing of exposure, as in the case of the nursing infant, and regarding more sensitive health endpoints relevant to an infant's and toddler's exposure. We anticipate that a suitable reference exposure level for total PBDEs may well be at or below the exposures estimated here for infants, if not other age groups as well. For example, the "average" infant exposure at 280 ng/kg$_{body\ wt}$·day is 10 times less than the RfD for the penta mixture that is based on hepatotoxicity, not developmental or neurological endpoints. However, maximum infant and toddler exposures could be >4100 and 1485 ng/kg$_{body\ wt}$·day, respectively, which are well within the RfD for penta. Thus, as Muir (*3*) points out, it is possible that population exposures at the 95th percentile may occur at levels that equal or surpass more sensitive developmentally or neurologically based toxicity thresholds. This reasoning provides the rationale for recent legislative and voluntary actions of political jurisdictions and industry to remove the penta and octa mixtures from the marketplace.

This study shows that dust is an important exposure pathway for PBDEs for all life stages. Dust is a likely contributor to elevated human milk concentrations that, through breast feeding, provide the greatest lifetime exposure for all life stages. Dust is also by far the greatest route of PBDE exposure to toddlers. What distinguishes these contaminants from legacy POPs is our exposure from predominantly indoor sources and notably consumer products that are common in homes and workplaces. These results suggest that relatively simple measures that can be taken now to reduce exposures to PBDEs and other organics (*57*) in house dust by minimizing the abundance of these products and their likelihood of emitting PBDEs into our homes, schools, and workplaces (e.g., removing old, crumbling foam furniture). As well, based on our knowledge of lead (*55*), removing dust by house cleaning is also a practical means of reducing one's exposure. The study also supports the need to restrict the use and release of PBDEs in order to minimize their ongoing distribution throughout the environment, at which point reducing exposure becomes much more difficult, as is the case with legacy POPs.

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## Note Added after ASAP Publication

Due to a production error, extraneous numbers appeared along the right margin of Figure 1 in the version published ASAP June 15, 2005. The corrected figure appears in the version published ASAP June 27, 2005.

## Supporting Information Available

Two tables. This material is available free of charge via the Internet at http://pubs.acs.org.

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